Odontogenic Orofacial Infections

Bertossi, Dario; Barone, Antonio; Iurlaro, Antonio; Marconcini, Simone; De Santis, Daniele; Finotti, Marco; Procacci, Pasquale

How to cite

BERTOSSI, Dario et al. Odontogenic Orofacial Infections. In: Journal of Craniofacial Surgery, 2017, vol. 28, n° 1, p. 197–202. doi: 10.1097/SCS.0000000000003250

This publication URL: https://archive-ouverte.unige.ch/unige:112391

Publication DOI: 10.1097/SCS.0000000000003250

Odontogenic Orofacial Infections

Dario Bertossi, MD,[*] Antonio Barone, MSc, DDS,[†‡] Antonio Iurlaro, DDS,[§] Simone Marconcini, DDS, PhD,[†‡] Daniele De Santis, MD, DDS,[*] Marco Finotti, MD, DDS,[||] and Pasquale Procacci, MD[*]

Abstract: Acute dental abscess is a frequent and sometimes underestimated disease of the oral cavity. The acute dental abscess usually occurs secondary to caries, trauma, or failed endodontic treatment. After the intact pulp chamber is opened, colonization of the root canals takes place with a variable set of anaerobic bacteria, which colonize the walls of the necrotic root canals forming a specialized mixed anaerobic biofilm. Asymptomatic necrosis is common. However, abscess formation occurs when these bacteria and their toxic products breach into the periapical tissues through the apical foramen and induce acute inflammation and pus formation. The main signs and symptoms of the acute dental abscess (often referred to as a periapical abscess or infection) are pain, swelling, erythema, and suppuration usually localized to the affected tooth, even if the abscess can eventually spread causing a severe odontogenic infection which is characterized by local and systemic involvement culminating in sepsis syndrome. The vast majority of dental abscesses respond to antibiotic treatment, however, in some patients surgical management of the infection may be indicated. In the present work, a retrospective analysis of the patients with dental orofacial infections referred to the Unit of Dentistry and Maxillofacial Surgery of the University of Verona from 1991 to 2011 has been performed.

Key Words: Odontogenic, orofacial infections, retrospective analysis

(*J Craniofac Surg* 2017;28: 197–202)

MATERIALS AND METHODS

This work is based on a systematic analysis of 690 medical records of patients hospitalized between 1991 and 2011 because of odontogenic orofacial infection.[1–6]

The activity was designed to get statistical and epidemiological data of interest. For this purpose the data were organized taking into account age, sex, and site of onset in order to obtain results easily comparable with those obtained from similar studies. Microbiological pattern and main features of therapy and hospitalization also were taken into account.

RESULTS

After an initial analysis of 690 patients treated over 16 years, it can be observed that 418 patients (60.58%) involved males and 372 females (39.42%) (Fig. 1). We analyzed the age of onset of the disease. Pediatric patients, under 17 years, represent the 8.41% of the total with 58 reported patients (Fig. 2). Through a division into age groups of treated patients, data described the higher prevalence of the disease in age from 11 to 60 years. The highest incidence is between 21 and 30 years (Fig. 3). The division between sexes within each age group provides a more detailed analysis. In this way, we observed that how the male/female ratio in the different intervals tends to increase, especially in the 4th, 5th, and 6th decades (Fig. 4).

The disease onset site has been certainly identified in 571 patients (82.75%) while in the remaining 119 (17.25%) surveys conducted by the operators did not identify a single causative factor but an association of several elements (Fig. 5).

We have analyzed what are the areas primarily involved in the disease onset. The sector most frequently responsible for disease is the posterior jaw, which was affected by abscesses 276 times on the left and 272 times on the right. Unlike that, the upper jaw is involved in forms that require patient hospitalization in a lower percentage of patients, and our data indicate that it was the site of infection in 140 patients in total (Fig. 6).

A further consideration is the detection of unique onset locations of the disease, which allows the classification in order of frequency of infection-related teeth. In our series, the tooth most frequently associated with odontogenic abscesses is 4.6, which has been associated the pathology in 76 patients. Then, the element 3.8 caused 74 patients, 3.6 and 4.8 caused 70 patients each, 3.7 was involved in 64 patients, and 4.7 in 53 patients. All other elements were involved in a much lower percentage of patients, or even have never been associated with disease (Fig. 7). Considering only the age from 21 to 30 years, the one with higher frequency of occurrence of the disease, we can confirm the general pattern: the back of the jaw is the more involved site. It is also evident in the simultaneous involvement of multiple elements (Fig. 8). We registered 18 patients originated from the deciduous dentition. Also in this subgroup inferior-posterior areas of the mouth have a higher involvement frequency (Fig. 9).

The microbiological pattern of the infections has also been studied. Most infections seem to be polymicrobial synergistic aerobic and obligate anaerobic pathogenic processes. By means of sterile collected specimen cultures, 7% of the patients contained exclusively aerobic bacteria and 20% of the specimens only anaerobic bacteria. On the other hand, 68% of the patients presented with combined aerobic/anaerobic bacteria, whereas in 5% no organisms were detected.

The graphic elaboration of recovery days by patient is represented by a Gaussian curve with peak at 4 days of

From the *Dental Clinic and Maxillofacial Surgery Department, University of Verona, Verona; †Department of Surgery, Medical, Molecular and Critical Area Pathology, University of Pisa, Pisa; ‡Tuscan Stomatologic Institute, Versilia General Hospital, Lido di Camaiore (LU); §Private Practice, Francavilla Fontana; and ||Private Practice, Padua, Italy.

Received January 17, 2016.
Accepted for publication September 3, 2016.

Address correspondence and reprint requests to Prof Dario Bertossi, MD, Associate Professor in the Maxillofacial Department, University of Verona, Policlininico GB Rossi, Piazzale L Scuro 37134, Verona, Italy; E-mail: dario.bertossi@univr.it

The authors report no conflicts of interest.

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000003250

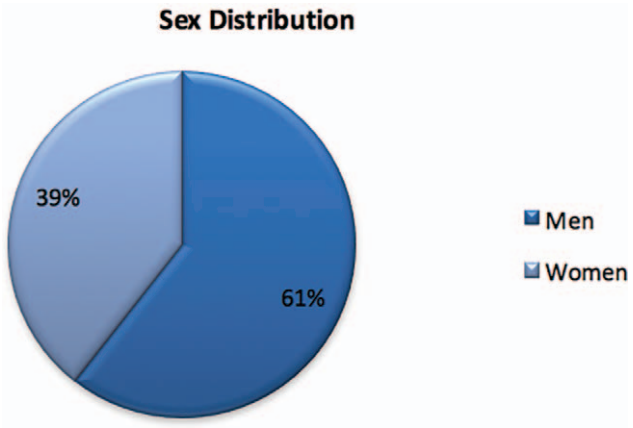

FIGURE 1. Sex distribution of the treated patients.

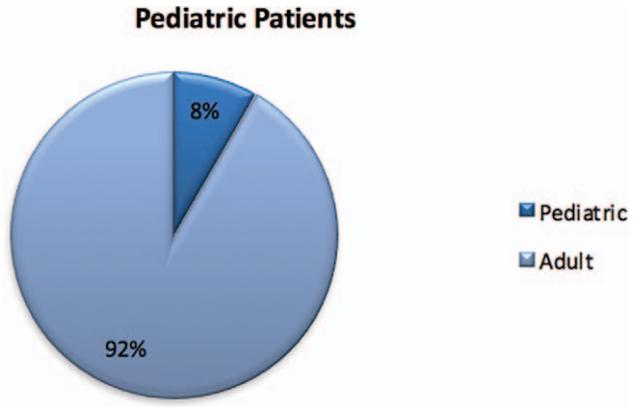

FIGURE 2. Incidence of pediatric patients.

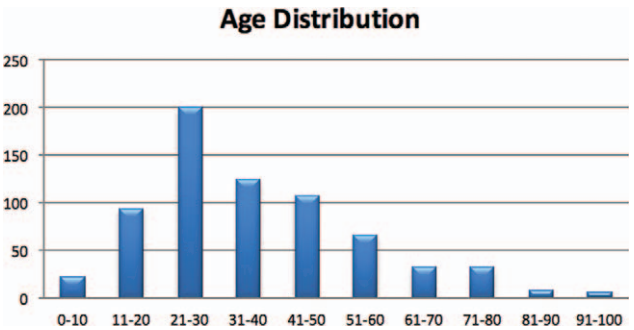

FIGURE 3. Age distribution of the treated patients.

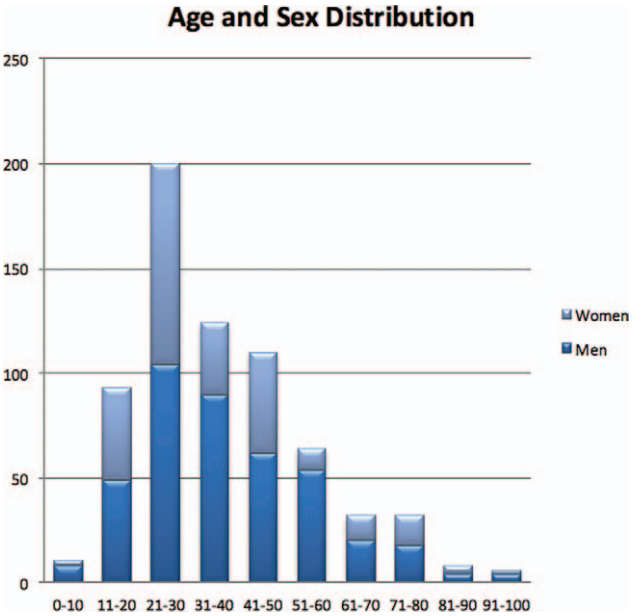

FIGURE 4. Combined age and sex distribution of the treated patients.

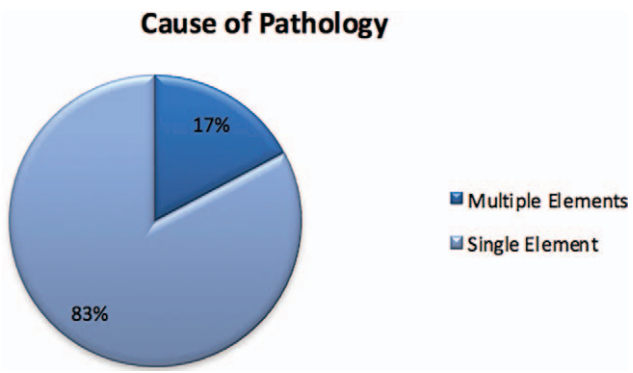

FIGURE 5. Cause of abscess single or multiple dental elements.

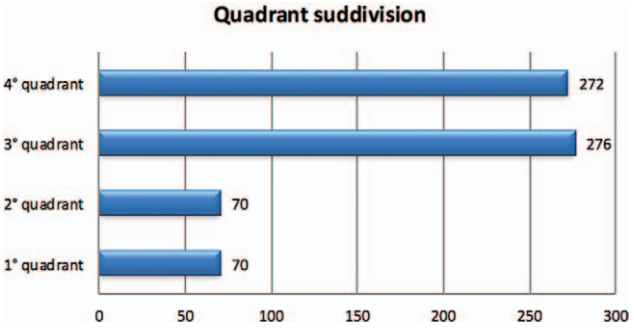

FIGURE 6. Localization of infections.

hospitalization. Patients hospitalized for 4 days were 187 (27.10%), for 5 days 140 (20.28%), and for 3 days (2.46%), whereas the patients with hospitalization for a period exceeding 10 days were 17 (2.46%) (Fig. 10). The increased length of hospitalization of the 17 patients hospitalized for more than 10 days is due to the clinical severity and therefore to the need for interventions that required a longer postoperative course (Fig. 11). A patient with mediastinitis required a 37-day hospitalization. It is important to point out that 5 patients resulted in a lateral cervical areas involvement with cervicofacial necrotizing fasciitis. In 3 patients, there were oral floor and suprahyoid area involvement that caused tongue dislocation and eventually Ludwig angina. Four patients presented the tonsillar pillar involvement. In 1 patient, the disease has spread to the parapharyngeal space. In another patient, it has spread to the temporal and infratemporal fossa. In 1 patient, it involved the orbital cavity and in 1 patient was observed multiple abscess formation with brain involvement.

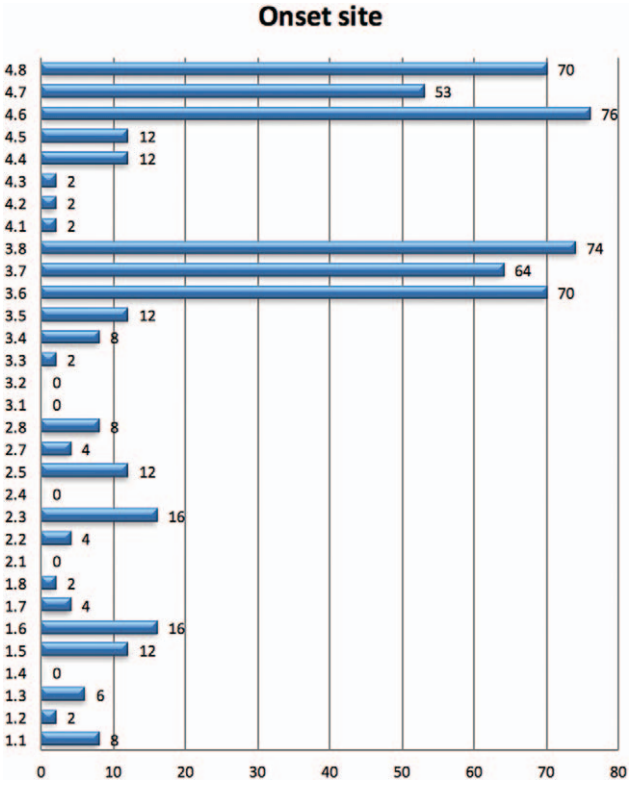

FIGURE 7. Onset site.

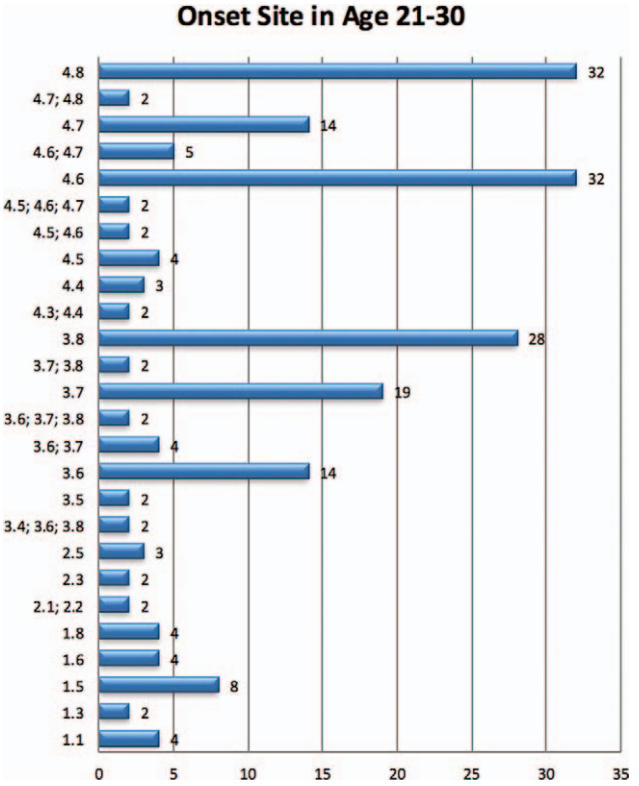

FIGURE 8. Onset site in age 21 to 30.

We have subdivided the disease treatment methods into 4 groups. The 61.45% of patients (424 patients) underwent abscess drainage associated with the extraction of involved elements, the alveolus remodeling, and an appropriate medical care. This includes the use of intravenous and/or oral antibiotics, hot-humid pack, appropriate exercises of mouth opening, non-steroid anti-inflammatory drugs, and the use of myorelaxants.

The 17.10% (118 patients) was subjected to pulpectomy and root canal medication, medical treatment and drainage of abscess often through the root canals. The 14.49% (100 patients) was treated with medical therapy and drainage of abscess, delaying dental treatment element. Finally, the 6.96% (48 patients) of patients were treated with medical therapy alone, the involved teeth were treated by private dentists (Fig. 12).

The 13.04% of patients (45 patients) needed general anesthesia for the complexity and type of intervention and the age. In the remaining 86.96% of patients, we used no anesthesia or regional anesthesia.

DISCUSSION

It is evident how the conditions that lay indication to the patient's hospitalization are those who involve the posterior of the jaw. This is due to an extreme danger of an untreated or incorrectly treated abscess in these parts that can cause the onset of complications which could endanger the patient's life.

The 3rd molar covers a major role in the frequency of pathology because it represents the cause in one-third of the patients that involves patients in the third of age. The dystopic or incomplete eruption of these elements induces a major susceptibility to the onset of decay and periodontal pathology and, then, of periradicular infections.

As far as microbiology is concerned, it is a common opinion that most odontogenic infections initially present as an aerobic bacteria-supported cellulitis. The cellulitis, when left untreated, enables the subsequent development of anaerobic infection. With the growing severity of the infection, a mixed flora takes over. Several proteolytic enzymes, endotoxins, and exotoxins are produced by the anaerobic share of the flora, with consequent tissue degradation, and immune reaction with abscess formation and pus. Eventually, if the host defense mechanisms succeed in limiting the infection as a well-circumscribed abscess, anaerobic bacteria only make it through.[7]

Aerobic gram-positive cocci like the α-hemolytic *Streptococcus viridans* group, often involved in subacute bacterial endocarditis, or the *Streptococcus milleri* group, often responsible of dental abscesses, appear to make up for the greatest part of early components of infections in the head and neck district. *Streptococcus*

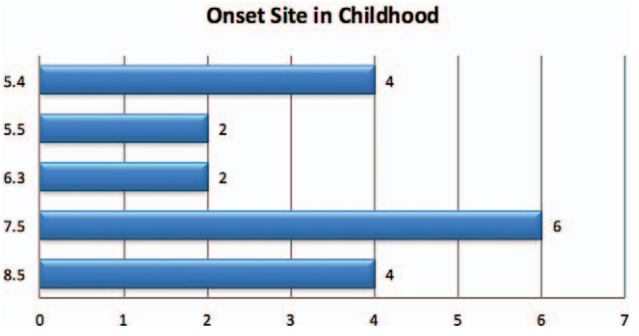

FIGURE 9. Onset in childhood.

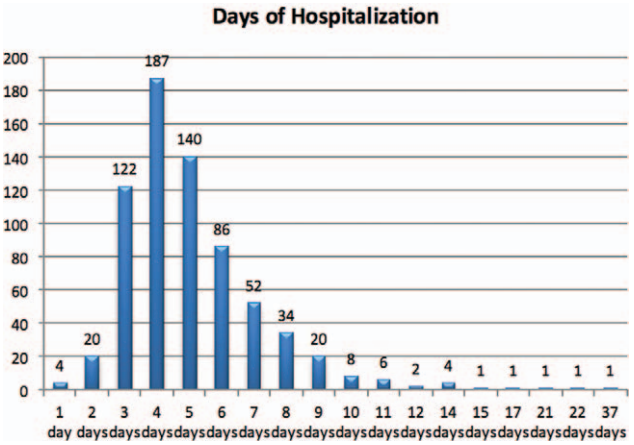

FIGURE 10. Days of hospitalization.

pyogenes, a β-hemolytic streptococcus, has recently had his role stressed in necrotizing fasciitis. *Staphylococcus aureus* is often found in toxic shock, endocarditis, chronic osteomyelitis, post-operative wound infections, and acute parotitis. *Hemophilus influenza*, on the other hand, is often implicated in sinusitis and infections of the ear.[8]

The subsequent anaerobic share of orofacial infections comprehends anaerobic gram-positive cocci and anaerobic gram-negative rods, such as the *Bacteroides* and *Fusobacterium* species. The mixed aerobic/anaerobic combination of *Streptococcus milleri* and *Fusobacterium* has been involved in life-threatening lateral and retropharyngeal descending infections, which may complicate into mediastinitis.[9]

Patients with abscesses that endanger patient's life are very rare, only 2.46% of treated patients needed hospitalization that lasted more than 10 days, and exclusively 1 patient needed to be referred to a thoracic surgery department.

Adequate antibiotic therapy must consider the microbiology and timing of the infection, antibiotic resistance, patient compliance, and cost. Early and outpatient infections seem to be adequately treated with therapy targeting aerobic streptococcal pathogens. In our experience, penicillins have been widely and successfully used.

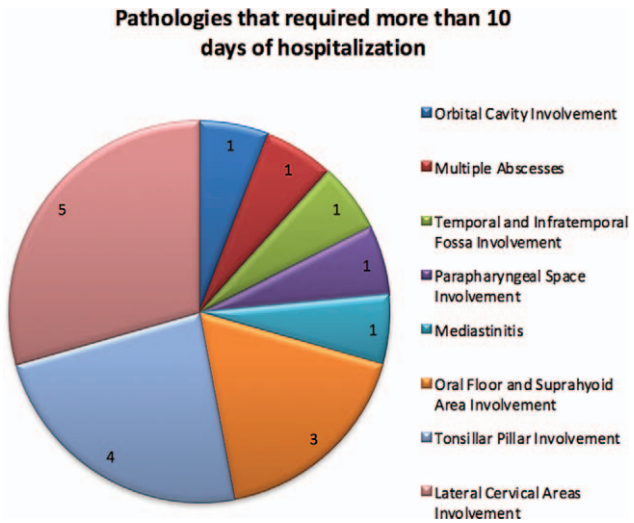

FIGURE 11. Pathologies with more than 10 days of hospitalization.

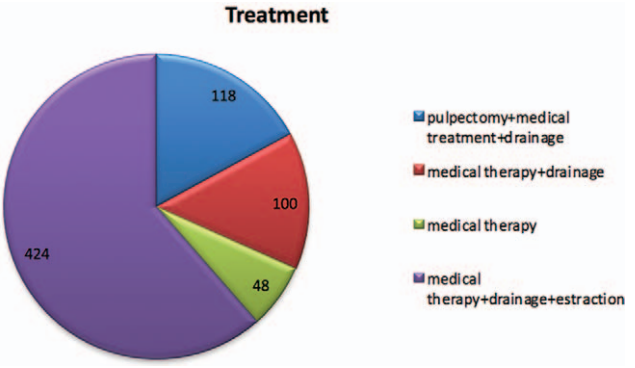

FIGURE 12. Treatment of pathology.

Penicillin-resistance rate among outpatient odontogenic infections remained low (5%), a data consistent with literature findings.[10] In patient with allergy to penicillin, erythromycin and macrolides proved to be a valid alternative. Cephalosporins also have been used, although less frequently because of the risk of cross-reaction with penicillins.

In more important infections, anaerobic gram-negative rods, which are often penicillin resistant (25–60%) must be targeted. Our antibiotic of choice in this patients was clindamycin, aiming to cover both streptococcus and anaerobes. Metronidazole is also valuable alternative against anaerobes, in association with ampicillin versus oral streptococci. Antibiotic management of nonresponsive outpatient odontogenic infections or more severe or life-threatening orofacial infections must be customized according to culture and sensitivity reports.[10]

The most severe and life-threatening complications of orofacial infections are Ludwig angina, cervicofacial necrotizing fasciitis, descending necrotizing mediastinitis, cavernous sinus thrombosis, and brain abscess. Despite their rarity, these complications present themselves still nowadays with remarkable morbidity and mortality.

Ludwig's angina is a form of severe diffuse cellulitis with acute onset and rapid spread resulting in bilateral affection of the submaxillary, sublingual and submental spaces, and woody-like swelling.[11] Due to the proximity of dental roots with these spaces, odontogenic infections are the most common etiology.[12] Ludwig angina can also be caused by other infective pathologies of the head and neck districts or by squamous cell carcinoma associated tissue necrosis.[13]

Systemic diseases causing severe impairment of health status may also be associated. Kurien et al[14] reported that in 52% of the adult patients, Ludwig angina was of dental origin and in 39%, the presence of predisposing systemic diseases could be identified; no predisposing illnesses could be detected in pediatric patients.[14]

The germ activity in Ludwig angina leads to significant muscular necrosis with late suppuration and locally aggressive behavior.[12]

Despite the strong impairment of the patient health status, there is no severe local discomfort. There is throat inflammatory swelling with clear edema of the oral pelvis.

Airway patency may be impaired; moreover, the infection can complicate with sepsis.

On the other hand, parapharyngeal spaces can also be involved with possible thoracic empyema. Ab ingestis pneumonia, meningitis, and vascular erosion have been cited as possible complications.[15]

Diagnosis is clinical. Computed tomography scan may help to precisely detect the diffusion of the infection.[16] Early antibiotic administration and prophylactic debridement of the spaces involved

are mandatory; airway patency must be preserved. Intravenous penicillin G, clindamycin, or metronidazole is the antibiotics recommended for use prior to obtaining culture and antibiogram results. Some authors also recommend the association of gentamycin.[10]

Mortality rates, due mainly to airway obstruction, vary depending on the different patient series studied, with figures that range from 4% to 60%.[11]

Cervicofacial necrotizing fasciitis is an uncommon soft tissue infection characterized by rapid spread. Large necrotic lesions and gas formation, located in the subcutaneous cell tissue and in the superficial fascia, can be observed. There is consequent myonecrosis and formation of petechiae.

When left untreated, cervicofacial necrotizing fasciitis evolves toward toxic shock and multiorgan failure.[17] Dental infections are the most frequent cause of the illness, although pharyngeal infections may also be associated.

Vasculopathies of various origin have been reported to be predisposing factors; however, the disease is observed also in healthy patients.[18,19]

The initial presentation of the disease can be misdiagnosed with a dental infection with soft tissue involvement until the progress of the pathology takes place, with resulting gangrene of the subcutaneous cell tissue and muscular aponeurosis, with swelling.

Although the onset pain is intense, later paraesthesia or even anesthesia may be observed because of nerve involvement. Skin may become affected, with poorly defined edges and almost black color. Gas and exudate formation may come to surface, from the 5th to the 9th day.

Later involvement of the neighboring tissues is possible, with local or systemic complications.[20] Health status is strongly impaired, with the signs of sepsis.

Computed tomography scan, detecting gas formation, is useful for the early diagnosis of this disease.[16]

Early treatment is strongly indicated and is based on broad-spectrum intravenous antibiotic therapy, surgical drainage of the lesion and in patient treatment of the responsible tooth. Drug therapy can be later modified according to the cultural examinations. Early surgical treatment is also mandatory, with incisions and drainages, in addition to debridement of soft tissues. Airway patency must be preserved through intubation or tracheotomy.[21]

Mortality rate (40%) associated with necrotizing fasciitis is high.[17] Related factors are the presence of associated diseases, timing of diagnosis, and the occurrence of shock.[21]

Mediastinitis is also uncommon and very severe. In mediastinitis, the parapharyngeal space and the retropharyngeal space are involved.

Once again, the spread of cellulitis is substained by both aerobic and anaerobic bacteria in synergy.

The semiology of mediastinitis presents with dysphagia, dyspnoea, stiff neck, and regurgitation. Swelling can be detected underneath the sternocleidomastoid muscle, with pain on palpation. As the infection makes its way to the mediastinum, retrosternal pain, dyspnoea, and nonproductive cough can be observed. Edema and crepitation in the upper thorax are present. The patient usually reports high fever with chills and extreme prostration.[22,23]

Chest x-ray reveals dislocation of the posterior wall of the pharynx on the lateral view and pneumomediastinum with broadening on the anteroposterior projection.[16] Systemic and local complications may take over and eventually lead to death.

Intravenous antibiotics at high doses targeting aerobic and anaerobic bacteria and cardiorespiratory support measures in the intensive care units are mandatory.

Even though surgery aiming to grant drainage of the infected space is unlikely to succeed, a transcervical approach has been recommended, from the anterior edge of the sternocleidomastoid muscle to the mediastinum through finger dissection via the pretracheal space, reducing the risk of vascular injuries. Drainage of the mediastinum is then pursued with suction drains and placing the patient in Trendelenburg position.[24]

Difficulty in making an early diagnosis, since dysphagia, dyspnoea, swelling of the neck, and crepitation are all late signs of the condition, makes mortality rates high.[22,23]

The 2 most severe intracranial complications of orofacial infections are cavernous sinus thrombosis and brain abscess. Odontogenic infections may involve the cavernous sinus through an anterior and posterior pathway, that is to say as a retrograde septic thrombophlebitis from the infraorbital space to the inferior ophthalmic vein through the inferior orbital fissure into the cavernous sinus, or via the pterygoid venous plexus to the inferior petrosal sinus into the cavernous sinus.[25]

The patient with cavernous sinus thrombosis may present with fever, headache, nausea, vomiting, supraorbital paresthesia, proptosis, photophobia ophthalmoplegia, chemosis, and ocular pain. Drainage of the infraorbital or infratemporal spaces and treatment of the responsible tooth are mandatory. High-dose intravenous antibiotics able to cross the blood brain barrier are indicated. The role for anticoagulation and steroids in these patients is not clear.

Very few brain abscesses can be ascribed to dental origin. Bacteria can reach[26] the brain as direct propagation or via the bloodstream. Brain abscesses present with fever, headache, and focal neurological deficits. Computed tomography scan and magnetic resonance imaging are pathognomonic. The microbiological pattern of the brain abscess reflects the original infective site.

Intravenous adequate antibiotic therapy is mandatory. Surgical options include aspiration or excision. Adequate monitoring must take place in neurological intensive care unit. Repeated aspiration may be necessary, but it may avoid residual neurological deficits associated with excision. Mortality rates is 20%.[26,27]

CONCLUSIONS

Most orofacial infections are routinary, innocuous lesions treated by all dental practitioners. Whenever the complications discussed above take place, adequate clinical examination, selective diagnostic imaging, appropriate antibiotic therapy, safe airway control, and time-effective surgery are the tools that the clinician has to master in order to preserve patient's life.

Future evaluations will also have to consider the socioeconomic aspects of population regarding to migration and to the significant presence of foreign patients (within the casuistry) who represent today, 15.94% of the patients (110 patients).

REFERENCES

1. LeJeune HB, Amedee RG. A review of odontogenic infections. *J La State Med Soc* 1994;146:239–241
2. Chavez De Paz LE. Redefining the persistent infection in root canals: possible role of biofilm communities. *J Endod* 2007;33:652–662
3. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. *Crit Rev Oral Biol Med* 2004;15:348–381
4. Flynn TR. The swollen face. Severe odontogenic infections. *Emerg Med Clin N Am* 2000;18:481–519
5. Gregoire C. How to manage odontogenic infections. *Todays FDA* 2010;22:37–39
6. Heimdahl A, Nord CE. Orofacial infections of odontogenic origin. *Scand J Infect Dis Suppl* 1983;39:86–91
7. Robertson D, Smith AJ. The microbiology of the acute dental abscess. *J Med Microbiol* 2009;58:155–162
8. Lopez-Piriz R, Aguilar L, Giménez MJ. Management of odontogenic infection of pulpal and periodontal origin. *Med Oral Patol Oral Cir Bucal* 2007;12:E154–E159

9. Goumas PD, Naxakis SS, Papavasiliou DA, et al. Periapical abscesses: causal bacteria and antibiotic sensitivity. *J Chemother* 1997;9:415–419
10. Levi ME, Eusterman VD. Oral infections and antibiotic therapy. *Otolaryngol Clin N Am* 2011;44:57–78
11. Saifeldeen K, Evans R. Ludwig's angina. *Emerg Med J* 2004;21: 242–243
12. Dugan MJ, Lazow SK, Beger JR. Thoracic empyema resulting from direct extension of Ludwig's angina: a case report. *J Oral Maxillofac Surg* 1998;56:968–971
13. Fischmann GE, Graham BS. Ludwig's angina resulting from the infection of an oral malignancy. *J Oral Maxillofac Surg* 1985;43: 795–796
14. Kurien M, Mathew J, Job A, et al. Ludwig's angina. *Clin Otolaryngol* 1997;22:263–265
15. Jiménez Y, Bagán JV, Murillo J, et al. Odontogenic infections: complications and systemic signs [in Italian]. *Med Oral Patol Oral Cir Bucal* 2004;9(suppl):S139–S147
16. Kim D, Lazow SK. Imaging head and neck infections. *Oral Maxillofac Surg Clin N Am* 2001;13:585–601
17. Balcerak RJ, Sisto JM, Bosack RC. Cervicofacial necrotizing fasciitis: report of three cases and literature review. *J Oral Maxillofac Surg* 1988;46:450–459
18. Rapoport Y, Himelfarb MZ, Zikk D, et al. Cervical necrotizing fasciitis of odontogenic origin. *Oral Surg Oral Med Oral Pathol* 1991;72:15–18
19. Chidzonga MM. Necrotizing fasciitis of the cervical region in an AIDS patient: report of a case. *J Oral Maxillofac Surg* 1996;54:638–640
20. Roberson JB, Harper JL, Jauch EC. Mortality associated with cervicofacial necrotizing fasciitis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82:264–267
21. Umeda M, Minamikawa T, Komatsubara H, et al. Necrotizing fasciitis caused by dental infection: a retrospective analysis of 9 cases and a review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003;95:283–290
22. Vieira F, Allen SM, Stocks RM, et al. Deep neck infection. *Otolaryngol Clin N Am* 2008;41:459–483
23. Rubin MM, Cozzi GM. Fatal necrotizing mediastinitis as a complication of an odontogenic infection. *J Oral Maxillofac Surg* 1987;45:529–533
24. Osborn TM, Assael LA, Bell RB. Deep space neck infection: principles of surgical management. *Oral Maxillofac Surg Clin N Am* 2008;20:353–365
25. Ogundiya DA, Keith DA, Mirowski J. Cavernous sinus thrombosis and blindness as complications of an odontogenic infection: report of a case and review of literature. *J Oral Maxillofac Surg* 1989;47:1317–1321
26. Corson MA, Postlethwaite KP, Seymour RA. Are dental infections a cause of brain abscess? Case report and review of the literature. *Oral Dis* 2001;7:61–65
27. Pedrazzoli M, Colletti G, Ferrari M, et al. Mesenchymal phosphaturic neoplasm in the maxillary sinus: a case report. *Int J Oral Maxillofac Surg* 2010;39:1027–1032